IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF NORTH CAROLINA
SOUTHERN DIVISION
NO. 7:23-CV-897

IN RE:

CAMP LEJEUNE WATER LITIGATION

This Document Relates To:

***McElhiney v. United States,* 7:23-cv-1368**
***Peterson v. United States,* 7:23-cv-1576**
***Rothchild v. United States,* 7:23-cv-858**
***Welch v. United States,* 7:23-cv-1503**
***Sparks v. United States,* 7:23-cv-682**

UNITED STATES' REPLY IN SUPPORT OF ITS MOTION TO EXCLUDE CERTAIN OPINIONS OF PLAINTIFFS' PARKINSON'S DISEASE EXPERTS, DRS. JASON CANNON, AMELIA BOEHME, MICHAEL FREEMAN, LUCIO COSTA, STEVEN BIRD, BRIANA DE MIRANDA, GARY MILLER, KRISTIN ANDRUSKA, HEIDI SCHWARZ, AND RICHARD BARBANO

("Unreliable Opinions That Perchloroethylene (PCE) Can Cause Parkinson's Disease")

INTRODUCTION

Missing from Plaintiffs' Opposition is any reference to *what their experts did to bridge the analytical gap* in the experts' opinions regarding perchloroethylene ("PCE") and Parkinson's disease ("PD").[1] Instead, Plaintiffs cite *new* literature in an attempt to show that their ten PD experts *could* have supported their opinions that PCE can cause PD. But this avoids an inconvenient fact: Plaintiffs' experts did not rely on, or even consider, this new literature with respect to the relationship between PCE and PD. Plaintiffs cite four animal studies to "outline the body of evidence [their experts] considered in applying their scientific judgment." D.E. 693 at 14, 19. However, only one expert cited to a single one of those studies. Further, Plaintiffs cite the Environmental Protection Agency's ("EPA") policy-making documents to bolster their experts' opinions. Yet, most of the experts never referred to such rule-making documents or cited the documents to support their opinions regarding PCE and PD. And, as a fallback, Plaintiffs continue to cite studies on Trichloroethylene ("TCE")[2] as support for their experts' opinions on PCE. But at deposition, the experts conceded that this theory is based on mere hypotheses and unproven biological processes. Ultimately, Plaintiffs still fail to demonstrate that their experts sufficiently explained how the literature on TCE is relevant to the relationship between PCE and PD. Plaintiffs do not get to build a makeshift bridge in their legal pleadings to cross the chasm their experts left empty.

Moreover, Plaintiffs attempt to transform an admissibility challenge into a dispute about scientific conclusions. But whether an expert has reliably applied his or her methods is a necessary inquiry under Federal Rule of Evidence 702. Plaintiffs' overreaching interpretation of the caselaw and misinterpretation of the United States' arguments does not transform the admissibility question before the Court into a dispute

[1] "Plaintiffs' Opposition" refers to Plaintiffs' Leadership Group's Response to the United States' Motion to Exclude Certain Opinions of Plaintiffs' Parkinson's Disease Experts, D.E. 693. Plaintiffs' Parkinson's diseases experts are Dr. Steven Bird, Dr. Gary Miller, Dr. Jason Cannon, Dr. Amelia Boehme, Dr. Briana De Miranda, Dr. Lucio Costa, Dr. Michael Freeman, Dr. Richard Barbano, Dr. Heidi Schwarz, and Dr. Kristin Andruska.

[2] The United States does not concede that the currently available evidence supports a causal association between TCE exposure at Camp Lejeune and PD. The focus of this Reply, however, is on Plaintiffs' experts' further improper extrapolation of their opinions on TCE to form opinions on PCE.

about scientific conclusions. The United States' motion presents a simple argument: Plaintiffs' experts failed to bridge the analytical gap in using literature on the relationship between TCE and PD to reach their conclusions about the relationship between PCE and PD. Therefore, their opinions on PCE are unreliable and should be excluded.

ARGUMENT

I. Plaintiffs Cannot Rely on New Sources that Their Experts Failed to Consider to Save The Experts' Unreliable Opinions.

As is more fully outlined in the United States' Opening Brief, D.E. 546 at 2–14, Plaintiffs' experts posit that the literature supporting their opinions on a causal relationship between TCE and PD can be seamlessly applied to the relationship between PCE and PD.[3] However, when questioned, the experts conceded that this theory is based on mere hypotheses and unproven biological processes. D.E. 546 at 25–26. Plaintiffs now endeavor to buttress their experts' opinions on PCE by referencing literature that their experts never considered. D.E. 693 at 10–20. But Plaintiffs ignore the fact that *the expert* must bridge the analytical gap in an expert disclosure; it cannot be cured by an attorney in a legal pleading. Post-hoc attempts in legal documents to explain the scientific connection that the expert failed to articulate are meaningless and insufficient to overcome the opinions' deficiencies under Rule 702. Moreover, the literature itself is unhelpful to Plaintiffs' argument because the studies answer questions entirely distinct from whether PCE can cause PD. Thus, Plaintiffs' experts' opinions on the relationship between PCE and PD *still* lack sufficient facts or data to support them.

A. EPA's TSCA Literature is Irrelevant to Plaintiffs' Experts' Opinions.

Plaintiffs' efforts to rely on new literature to support their experts' faulty opinions begin by pointing

[3] Plaintiffs assert that Drs. De Miranda, Costa, Barbano, Schwarz, and Andruska will not be offering general causation opinions on PCE. *See* D.E. 693 at 13 n.4. However, as outlined in the Opening Brief, the United States maintains that these experts offered opinions on PCE and PD. *See generally* Costa Rep. at 23 (JA Ex. 127, D.E. 467-10) ("[A] causal link between PCE and PD also exists, although based on less evidence."). Further, in their Brief in Opposition to the United States' Motion to Exclude Certain Opinions of Gary Miller, Lucio Costa, and Richard Barbano, Plaintiffs contradict themselves by stating that "Dr. Barbano also offers the general causation opinion that TCE and PCE are at least as likely as not to cause PD." D.E. 682 at 21. Pursuant to the Court's July 22, 2025 Order, D.E. 444, general causation opinions from the Phase III experts are untimely. Accordingly, any general causation opinions on PCE from these experts should be excluded for the reasons explained in the United States' Opening Brief and this Reply.

to EPA's rulemaking under the Toxic Substances Control Act ("TSCA") regarding PCE and TCE. In doing so, Plaintiffs overlook three simple facts: (1) their experts did not rely on the TSCA rule-making documents to reach their opinions regarding PCE and PD; (2) the TSCA rule did not determine that PCE causes PD; and (3) the TSCA rule is not specific to the levels of PCE in the water at Camp Lejeune nor the routes of exposure at issue in this litigation.[4]

First and foremost, Plaintiffs' experts did not rely on EPA's TSCA rule-making documents in forming their opinions. Rule 26 is clear: the expert must provide "a complete statement of all opinions that that witness will express and the basis and reasons for them" and must disclose "the facts and data considered by the witness" in forming the expert opinions. Fed. R. Civ. P. 26(a)(2)(B)(i)–(ii). Plaintiffs cannot rely on sources that were not included in the experts' disclosures or referenced in deposition testimony to defeat a *Daubert* challenge. *See Hirchak v. W.W. Grainger, Inc.*, 980 F.3d 605, 609 (8th Cir. 2020) (materials not considered by the expert cannot "rescue an expert opinion from inadmissibility by filling its analytical gaps"). Most of Plaintiffs' experts did not cite EPA's final rule, nor the underlying risk evaluation for PCE that was published in 2020. EPA 2020 PCE Risk Eval. (JA Ex. 198, D.E. 473-6). Rather, they make vague references to a press release. *See, e.g.,* Barbano Rep. (Peterson) at 14 (JA Ex. 529, D.E. 502-6). In fact, at his deposition, Dr. Gary Miller testified that he never read the PCE TSCA rule; he had merely heard about it. Miller GC Dep. Tr. at 284:6–7 (JA Ex. 171, D.E. 470-10). The issue before the Court is not whether Plaintiffs can identify documents that complement their experts' reports after the fact. The issue is whether the experts themselves adequately explained and reliably supported their opinions to be admitted under Rule 702. Here, they did not.

Moreover, EPA's review of the literature for its regulatory determinations does not necessarily meet the standard of reliability for causation opinions in a tort case. Courts recognize "a distinction between evaluations made by regulatory agencies and standard of causation necessary to show tort liability." *Yates*

[4] EPA is reconsidering its rulemaking on PCE due to implementation issues and has requested comments on whether alternative measures could be taken. *See Perchloroethylene (PCE); Regulation Under the Toxic Substances Control Act (TSCA); Request for Comment*, 90 Fed. Reg. 35858 (July 30, 2025).

v. Ford Motor Co., 113 F. Supp. 3d 841, 847 (E.D.N.C. 2015) ("The agencies' threshold of proof is reasonably lower than that appropriate in tort law, which traditionally makes more particularized inquiries into cause and effect and requires a plaintiff to prove that it is more likely than not that another individual has caused him or her harm.") (internal citations omitted). Thus, to say that the rulemaking "complement[s] and confirm[s] the reliability" of Plaintiffs' experts' opinions is factually incorrect. *See* D.E. 693 at 11.

Second, had Plaintiffs' experts relied upon EPA's rulemaking documents, they would have recognized that EPA did not conclude that PCE can cause PD. Even under the lesser regulatory standard that EPA applied for TSCA, EPA did not conclude that PCE can cause PD. *See* Perchloroethylene (PCE); Regulation Under the Toxic Substances Control Act (TSCA), 89 Fed. Reg. 103560, 103562 (Dec. 18, 2024) (to be codified at 40 C.F.R. pt. 751) (recognizing adverse impacts as a result of chronic exposure including kidney and liver effects, immune system toxicity, reproductive toxicity, developmental toxicity, cancer, impaired visual and cognitive function, and diminished color discrimination). As such, Plaintiffs' experts could not have relied on the rulemaking to support their opinions regarding PCE and PD even if they had considered it. As a related matter, Plaintiffs' current reliance on EPA's TSCA rulemaking raises questions about how their experts interpreted the studies that they actually relied upon in their reports. For example, Plaintiffs' experts rely heavily on Goldman (2012) to support their opinions; this is the *only* epidemiological study that Plaintiffs' experts identified that considered PCE exposure and its potential association to PD. Goldman 2012 (JA Ex. 252, D.E. 480-12). In EPA's TSCA risk evaluation for PCE, EPA reviewed Goldman (2012) and concluded that the association between self-reported PCE exposure and PD was "was not statistically significant *and highly unstable* (OR: 10.5; 95% CI = 0.97, 113)," given the extremely wide confidence interval. EPA 2020 PCE Risk Eval. at 295 (JA Ex. 198, D.E. 473-6) (emphasis added). This demonstrates that Plaintiffs' experts' opinions regarding PCE and PD are on shaky ground, even when viewed through the literature that Plaintiffs' counsel now insists supports the experts' theories.

Further, EPA's TSCA evaluations do not even seek to answer the question of general causation for the environmental levels of PCE that may have been present at Camp Lejeune. Under TSCA Section 6(b), EPA conducts "risk evaluations to determine whether a chemical substance presents unreasonable risk of

injury to health or the environment, *under the conditions of use*, without consideration of costs or other non-risk factors, including an unreasonable risk to potentially exposed or susceptible subpopulations, identified as relevant to the risk evaluation." EPA 2020 PCE Risk Eval. at 34 (JA Ex. 198, D.E. 473-6) (citing 15 U.S.C. § 2605(b)) (emphasis added). The "conditions of use" that EPA considers include only circumstances ''under which a chemical substance is intended, known, or reasonably foreseen to be manufactured, processed, distributed in commerce, used, or disposed of." 15 U.S.C. § 2602(4). EPA is not tasked with considering the effects of chemicals in drinking water, as it is not a defined condition of use. Nor is EPA tasked with answering the question of whether the level of PCE contamination at Camp Lejeune can cause PD, which was the question for experts in this case. Accordingly, EPA's TSCA rulemaking provides no basis to support the reliability of conclusions of Plaintiffs' experts regarding PCE and PD.

B. The Animal Studies Cited in Plaintiffs' Opposition are Irrelevant to Plaintiffs' Experts' Opinions.

In another after-the-fact attempt to save their experts' unreliable opinions on PCE and PD, Plaintiffs' attorneys cite four animal studies. Only one of Plaintiffs' experts cited *any* of those studies, and that expert only cited *one* of them. The remaining nine experts did not consider any of the four animal studies that are detailed in Plaintiffs' Opposition. Given that the experts did not consider the studies, the studies cannot retroactively supply support for the experts' opinions. Regardless, even if Plaintiffs' experts had considered the studies, Plaintiffs' argument misses the mark.

Of the three never-before-cited studies, none of them demonstrates how the experts could reach their opinions regarding a relationship between PCE and PD.[5] Dr. Jason Cannon is the sole expert to cite any of the four studies that are listed in Plaintiffs' Opposition. He cited Mattsson (1993), D.E. 693-12, but provided merely one sentence in his report about the study about potential effects in the visual cortex of rats. Cannon Rep. at 26 (JA Ex. 123, D.E. 467-6). He did not explain how Mattsson (1993)—a study that

[5] Plaintiffs also include two studies on TCE, only one of which their experts considered: Gash and Rosengren. D.E. 693-13; 693-16. Just as Plaintiffs' experts have not explained in their reports how one can jump from TCE literature to conclusions on PCE, Plaintiffs fail to provide any explanation regarding how these studies support the analytical leap.

never mentions PD—could support a conclusion that PCE can cause PD. Yet, Plaintiffs now assert that Mattsson (1993) supports all of their experts' opinions on the relationship between PCE and PD. D.E. 693 at 19. While the four studies, including the three that no expert cited, focus on the neurotoxicity of PCE, Plaintiffs cannot demonstrate how their experts used those studies to draw a conclusion that PCE can cause PD at the levels of contamination at Camp Lejeune. Not only would this require the experts to consider the studies and describe their relevance to Camp Lejeune PCE contamination levels, which they did not do, but it would also require them to explain how the toxicological studies involving laboratory animals are applicable to humans. Courts routinely recognize the limitations of animal studies in drawing conclusions to be extrapolated to humans. *See, e.g.*, *Reference Manual on Scientific Evidence* 563 (3d ed. 2011) (hereinafter "*RMSE*"). As such, experts must explain how the results of animal studies could be extrapolated to humans to reach conclusions regarding general causation in humans. *See Dunn v. Sandoz Pharms. Corp.*, 275 F. Supp. 2d 672, 683 (M.D.N.C. 2003) (excluding an expert for failure to address how animal studies could form the basis of his general causation opinions). Here, Plaintiffs' experts (with one exception) did not consider any of these studies, let alone venture to explain how these animal studies could help them reach their opinions regarding whether PCE can cause PD in humans.

II. Plaintiffs Cannot Use the Referenced Epidemiology Studies to Provide a Sufficient and Reliable Basis for Their Experts' Opinions on PCE and Parkinson's Disease.

As explained in the United States' Opening Motion, the epidemiological studies that Plaintiffs' experts cite are insufficient to provide a basis for an expert to reach the opinion that PCE can cause PD because the studies are not specific to PCE. *See* D.E. 546 at 24–27. As Plaintiffs' experts explained, the lack of studies on PCE and PD is due to a lack of interest by academics. Miller GC Rep. at 18 (JA Ex. 132, D.E. 467-15) ("Academic researchers have focused on TCE and that is why there are more papers on TCE."); Cannon Rep. at 22 (JA Ex. 123, D.E. 467-6) ("To date, PCE simply has not been investigated as a PD risk factor as thoroughly or rigorously as TCE."); Costa Rep. at 21 (JA Ex. 127, D.E. 467-10) ("Thus, the contribution of exposure to PCE to PD, while less studied than that of TCE, cannot be discounted."). Yet, notably missing from Plaintiffs' explanation of the epidemiology studies is any reference to how their

experts could reach their opinions on PCE without sufficient supporting epidemiological literature on PCE and PD. *Small v. WellDyne, Inc.*, 927 F.3d 169, 177 (4th Cir. 2019) (citing *Daubert*, 509 U.S. at 590) ("[I]t is not enough for an expert to rely on his subjective belief.").

Only one study listed considered by Plaintiffs' experts analyzed PCE exposure and PD without the presence of other chemicals: Goldman (2012). (JA Ex. 252, D.E. 480-14). However, Goldman (2012) did not find a statistically significant odds ratio for individuals exposed to PD and PCE. *Id.* at 779. As EPA characterized it, the association "was not statistically significant *and highly unstable*." EPA 2020 PCE Risk Eval. at 295 (JA Ex. 198, D.E. 473-6) (emphasis added). One study that yielded a statistically insignificant finding with an extremely wide confidence interval cannot supply a reliable basis for the experts' opinions.

Plaintiffs cite four other epidemiological studies, but none of those studies analyzed PCE exposure without the presence of co-exposures with other chemicals. As the Supreme Court explained in *General Electric Co. v. Joiner*, 522 U.S. 136, 145 (1997), an expert's reliance on a study in which the participants were exposed to multiple chemicals was "of no help" in supporting the expert's opinion. The same is true here. Plaintiffs rely on four studies analyzing the Camp Lejeune population: Bove 2014 Mort. Study - Civ. (JA Ex. 189, D.E. 472-10), ATSDR 2018 Morbidity Study (JA Ex. 184, D.E. 472-5), Goldman 2023 (JA Ex. 253, D.E. 480-13), and Goldman 2024 (JA Ex. 254, D.E. 480-14). Because individuals at Camp Lejeune were potentially exposed to various contaminants such as TCE, PCE, vinyl chloride, and benzene, the studies cannot isolate the effect of any one chemical to properly understand the health effects of that individual chemical. As Dr. Cannon recognized in his report that, "contributions to PD risk from PCE cannot be fully separated in [Goldman 2023]." Cannon Rep. at 16 (JA Ex. 123, D.E. 467-6). Accordingly, the experts' jump from these studies to their opinions are based on an insufficient foundation.

Compounding the issue of extrapolating results from co-exposures, these studies have further limitations that Plaintiffs' experts chose not to highlight in their reports and Plaintiffs' counsel ignored in their Opposition. For example, Goldman (2024) did not analyze whether contamination at Camp Lejeune could cause PD. Rather, the study analyzed "whether PD progression is faster in individuals exposed to VOCs in water at Camp Lejeune." Goldman 2024 at 1 (JA Ex. 254, D.E. 480-14). Additionally, ATSDR

2018 is riddled with limitations, so much so that the lead epidemiologist, Dr. Frank Bove, chose not to submit it to any peer review journals because he recognized that the study had extreme limitations.[6] D.E. 540-2, Oct. 17, 2024 Bove. Dep. Tr. at 296:5-12. Yet, Plaintiffs' experts did not address these issues, thus failing to bridge the gap between the literature's flaws and their opinions.

III. Plaintiffs Cannot Sanitize Their Experts' Concessions Through Attorney Explanations.

Plaintiffs' counsel also seek to bolster their experts' opinions on the similarities between PCE and TCE to bridge the gap in their experts' opinions on PCE and PD. But nothing here fills the gaping holes in the text of the experts' reports and their concessions in deposition. "In a toxic tort case, the extrapolation or leap from one chemical to another must be reasonable and scientifically valid." *Aldridge v. Goodyear Tire & Rubber Co.*, 34 F. Supp. 2d 1010, 1023–24 (D. Md. 1999) (citations omitted), *vacated and remanded on other grounds*, 223 F.3d 263 (4th Cir. 2000). The leap of Plaintiffs' experts from TCE to PCE is suspended on speculation in the hope for favorable results from future studies that may confirm their unproven hypothesis that PCE can cause PD. Because the hypothesis is founded on speculation it cannot support an admissible opinion under Rule 702. *See Small v. WellDyne, Inc.*, 927 F.3d 169, 177 (4th Cir. 2019) (citing *Daubert*, 509 U.S. at 590) ("[I]t is not enough for an expert to rely on his subjective belief.").

Nothing in Plaintiffs' Opposition dispels their own experts' concessions and equivocations relating to the hypothesis. For example, when asked at deposition about the idea that a common metabolite between TCE and PCE could cause PD, Dr. Miller explained, "I'm not convinced it's TaClo and this is why my own laboratory has been looking at these metabolites with more modern techniques to see if we can figure out what these metabolites are. . . . I'm not convinced that is what the molecule is." Miller GC Dep. Tr. at 176:23–177:11 (JA Ex. 171, D.E. 470-10). Likewise, Dr. Briana De Miranda explained that the similarity

[6] Isolating the effect of PCE is important in this case because the predominant contaminant at Tarawa Terrace was PCE, while the main contaminant at Hadnot Point was TCE. For example, the flawed ATSDR 2018 study tried to isolate the effect of PCE on PD and the results failed to find any statistically significant associations between PCE and PD. ATSDR 2018 Morbidity Study (JA Ex. 184, D.E. 472-5). Overall, the incidence of PD in Marines was less in the Camp Lejeune cohort than in the Camp Pendleton cohort (OR 0.89 (95% CI 0.51, 1.55)). *Id.* at 74, Table 6. And the dose-response analyses for PCE and PD in Marines failed to show a monotonic dose response trend over three levels of exposure that would support an inference of causation. *Id.* at 78, Table 8.

between TCE and PCE in metabolic processes was something that "could be predicted" but conceded that it has not been proven because there was no "peer-reviewed published peer-review data on that." De Miranda Rep. at 11 (JA Ex. 129, D.E. 467-12); Dep. Tr. at 208:1–22 (JA Ex. 170, D.E. 470-9). Plaintiffs' experts' leap remains a "guilt-by-association" inference in analogizing PCE to TCE—the exact type of analytical leap that courts across the country have rejected. [7] *Caraker v. Sandoz Pharms. Corp*, 188 F. Supp. 2d 1026, 1038 (S.D. Ill. 2001). [8]

IV. Plaintiffs Cannot Establish a Predicate Association for PCE and PD.

Plaintiffs misinterpret the United States' arguments on predicate association to claim that the United States is demanding statistical significance throughout the experts' analysis. As Plaintiffs explained, their experts purported to apply the Bradford Hill criteria to evaluate whether an association establishes a causal relationship. D.E. 693 at 23; *see also RMSE* at 597–600. However, Plaintiffs overlook the first step of the Bradford Hill analysis: identify a clear-cut association. *RMSE* at 598–99 (finding that consideration of the remaining criteria of Bradford Hill is employed "only *after* a study finds an association to determine whether that association reflects a true causal relationship") (emphasis in original). The United States correctly represented the holding in *In re Lipitor* as requiring a statistically significant association or *some other analysis showing that the association is beyond the play of chance* to meet the predicate association requirement of Bradford Hill. *See In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs. & Prods. Liab. Litig. (No II) MDL 2502*, 892 F.3d 624, 640 (4th Cir. 2018) (affirming exclusion of expert because he "did not reliably apply the epidemiological/Bradford Hill method, which requires at the outset a statistically significant association before applying the Bradford Hill factors . . . ."). After that, the expert may proceed to analyze the association for causation by considering the Bradford Hill criteria.

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[7] Similarly, Plaintiffs attempt to rely on citations to the expert report of Dr. Amelia Boehme to justify their experts' opinions. However, Dr. Boehme is not a toxicologist and, as she admits in her deposition, not qualified to opine on any purported molecular similarities between TCE and PCE. Boehme Dep. Tr. at 132:23–133:23; 145:10–146:21 (JA Ex. 167, D.E. 470-6).

[8] For further discussion and direct testimony demonstrating Plaintiffs' experts' failure to bridge the analytical gap regarding the molecular structure of the chemicals and unproven common metabolites, *see* United States' Opening Brief, D.E. 546 at 16–24.

To be clear, the Fourth Circuit held that a district court properly excluded the plaintiff's expert for failing to identify a statistically significant association, *in the specific context of step one of the Bradford Hill criteria. Id.* Accordingly, Plaintiffs' fear that experts will never be permitted to consider results that are not statistically significant will not come to pass under *In re Lipitor*. After meeting the requirements of step one of Bradford Hill, experts may move to step two, where they must reliably apply the criteria to render an admissible Rule 702 causation opinion. The failure of Plaintiffs' experts to meet this clear threshold is due to a lack of supporting scientific evidence, not an overreach on the part of the United States.

Here, Plaintiffs' experts did not meet the predicate association requirement with respect to PCE because the sole study that analyzed PCE exposure on its own did not produce results that were statistically significant or otherwise shown to be beyond the play of chance. Goldman 2012 at 779 (JA Ex. 252, D.E. 480-12). Moreover, the confidence interval was so wide that EPA characterized the association as "unstable." *See* EPA 2020 PCE Risk Eval. at 295 (JA Ex. 198, D.E. 473-6). None of Plaintiffs' experts offered an explanation as to how such a result was more than chance, as required to employ the Bradford Hill criteria. Recognizing that the studies did not support their theory, Plaintiffs' experts chose to perform the analysis for TCE and summarily proclaim that the conclusions apply to PCE as well.

Finally, the United States' motion does not ask the Court to make a scientific determination. Rather, the Court need only examine whether Plaintiffs' experts reliably applied scientific knowledge to sufficient scientific data in order to conclude that PCE can cause PD. *See* Fed. R. Evid. 702 committee note to 2023 amendment (stating that critical questions of the sufficiency of an expert's basis, and the application of the expert's methodology, are proper questions of admissibility under Rules 702 and 104(a) and not weight). Accordingly, the issues before the Court fall squarely within its analysis under Rule 702.

CONCLUSION

For the reasons stated above and in the Opening Brief, the United States respectfully requests that this Court grant this Motion and exclude Plaintiffs' experts' opinions that PCE can cause Parkinson's Disease.

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Respectfully submitted,

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CERTIFICATE OF SERVICE

I hereby certify that on December 12, 2025, I electronically filed the foregoing using the Court's Electronic Case Filing system, which will send notice to all counsel of record.

/s/ Elizbeth K. Platt
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